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Amendments to the Claims

This listing of claims will replace all prior versions, and listings, of claims in the application;

Listing of Claims

- 1. (Twice Amended) A transgenic [rodent] mouse whose cells contain a DNA sequence comprising:
 - (a) a nerve tissue specific promoter operatively linked to a DNA sequence which encodes amyloid-beta peptide binding alcohol dehydrogenase (ABAD), and
 - (b) a nerve tissue specific promoter operatively linked to a DNA sequence encoding each of mutant human amyloid precursor proteins hAPP695, hAPP751, and hAPP770 bearing mutations linked to familial Alzheimer's disease in humans,

wherein said [rodent] mouse exhibits at least one phenotype from the group consisting of: reduced basal synaptic transmission; inhibited synaptic plasticity; increased neuronal stress; elevated 4-hydroxynonenal in cerebral cortex; increased heme oxygenase type I in cerebral cortex; decreased microtubule-associated protein 2 in cerebral cortex; and increased levels of activated caspase 3 antigen in cortical neurons.

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- 2. (Twice Amended) The transgenic [rodent] mouse of claim 1, wherein the promoter of both element (a) and (b) is platelet derived growth factor (PDGF)-B-chain promoter.
- 3. (Canceled)
- 4. (Canceled)
- 5. (Twice Amended) A method for evaluating in a transgenic [rodent] mouse the potential therapeutic effect of an agent for treating Alzheimer's disease in a human, which comprises:
 - (a) providing an agent to a transgenic [rodent] mouse whose cells comprise
 - (i) a nerve tissue specific promoter operatively linked to a DNA sequence which encodes amyloidbeta peptide binding alcohol dehydrogenase (ABAD), and
 - (ii) a nerve tissue specific promoter operatively linked to a DNA sequence encoding each of mutant human amyloid precursor proteins hAPP695, hAPP751 and hAPP 770 bearing mutations linked to familial Alzheimer's disease,
 - (b) determining the therapeutic effect of the agent on the transgenic [rodent] mouse by monitoring basal synaptic

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transmission or synaptic plasticity, wherein an increase in basal synaptic transmission or synaptic plasticity indicates that the agent would have a potential therapeutic effect on Alzheimer's disease in humans.

- 6. (Original) The method of claim 5, wherein the promoter of both element (a) and (b) is platelet derived growth factor (PDGF) b-promoter.
- 7. (Canceled)
- 8. (Canceled)